WEST Search History

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DATE: Monday, July 30, 2007

Hide?	Set Name	Query	Hit Count
	DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ		
☐	L20	PLD and peptide complex	19
☐	L19	PLD-2 and peptide complex	0
☐	L18	PLD-2 antibody and peptide complex	0
☐	L17	PLD-2 antibody peptide complex	0
☐	L16	beta-actin and peptide complex	331
☐	L15	beta-actin near actin	10917
☐	L14	beta-actin near anti-actin	0
☐	L13	beta-actin near actin	10917
☐	L12	beta-actin with actin	10917
☐	L11	beta-actin and actin	10917
☐	L10	beta-actin and actic	1
☐	L9	L7 and PLD-2	0
☐	L8	L7 and PLD-2 antibody	0
☐	L7	beta-actin	10917
☐	L6	anti-phospholipase D and beta-actin	0
☐	L5	anti-phospholipase D	2
☐	L4	phospholipase D antibody	1
☐	L3	PLD-2 antibody	0
☐	L2	PLD-2 antibody and beta-actin	0
☐	L1	PLD-2 antibody and actin	0

END OF SEARCH HISTORY

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=> file medline hcaplus biosis biotechds scisearch
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

FILE 'MEDLINE' ENTERED AT 15:56:21 ON 30 JUL 2007

FILE 'HCAPLUS' ENTERED AT 15:56:21 ON 30 JUL 2007
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FILE 'SCISEARCH' ENTERED AT 15:56:21 ON 30 JUL 2007
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=> s phospholipase and actin
L1        3312 PHOSPHOLIPASE AND ACTIN

=> dup rem l1
PROCESSING IS APPROXIMATELY  43% COMPLETE FOR L1
PROCESSING COMPLETED FOR L1
L2         1712 DUP REM L1 (1600 DUPLICATES REMOVED)

=> s l2 and antibody
L3          145 L2 AND ANTIBODY

=> s l2 and phospholipase antibody
L4            0 L2 AND PHOSPHOLIPASE ANTIBODY

=> s l2 and pld antibody
L5            0 L2 AND PLD ANTIBODY

=> s l2 and pld antibody
L6            0 L2 AND PLD ANTIBODY

=> s l2 and pld-antibody
L7            0 L2 AND PLD-ANTIBODY

=> s l2 and phospholipase-antibody
L8            0 L2 AND PHOSPHOLIPASE-ANTIBODY

=> s actin and phospholipase-antibody
L9            0 ACTIN AND PHOSPHOLIPASE-ANTIBODY

=> s actin and anti-phospholipase-D
L10           0 ACTIN AND ANTI-PHOSPHOLIPASE-D

=> s  anti-PLD2 and actin
L11           0 ANTI-PLD2 AND ACTIN

=> registry
REGISTRY IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> file registry
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
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L20          1 L19 AND BETA-ACTIN

=> d l20 ibib ab

L20  ANSWER 1 OF 1  HCAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         2006:795802  HCAPLUS
DOCUMENT NUMBER:          145:246606
TITLE:                    Marker genes for the diagnosis of chronic fatigue
                          syndrome by gene expression profiling
INVENTOR(S):              Gow, John; Chaudhuri, Abhijit
PATENT ASSIGNEE(S):       The University Court of the University of Glasgow, UK
SOURCE:                   PCT Int. Appl., 169pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.          DATE
     ---------------      ----   --------    -------------------      --------
     WO 2006082390         A1    20060810    WO 2006-GB332            20060201
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR,
             KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX,
             MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE,
             SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC,
             VN, YU, ZA, ZM, ZW
         RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
             IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ,
             CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH,
             GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM
PRIORITY APPLN. INFO.:                          GB 2005-2042        A  20050201
AB   Genes that show changes in levels of expression in chronic fatigue
     syndrome (myalgic encephalitis) are identified for use in the diagnosis of
     the disease and in its treatment.  These genes include those encoding
     defensin .alpha.1, Hb .gamma., CXCR4, tubulin .beta.1, serine/threonine
     kinase 17B, HLA-DR.beta.4, and prostaglandin D2 synthase.  There is a
     relatively small set of genes, identified as a hub set, that show changes
     in expression that result in changes in levels of expression of a no. of
     dependent or network genes.  The genes identified provide objective
     disease markers that may be used in diagnostic tests to support the
     diagnosis of CFS/ME or for monitoring the effectiveness of therapy.  They
     also provide a rational basis for classifying CFS/ME patients according to
     the biochem. lesion underlying their symptoms and enable provision of
     appropriate targeted therapies.
REFERENCE COUNT:          7     THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS
                                RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT


=> s l19 and actin
L21          17 L19 AND ACTIN

=> dup rem l21
PROCESSING COMPLETED FOR L21
L22          13 DUP REM L21 (4 DUPLICATES REMOVED)

=> d l22 1-12 ibib ab

L22  ANSWER 1 OF 13  HCAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         2006:795802  HCAPLUS
DOCUMENT NUMBER:          145:246606
TITLE:                    Marker genes for the diagnosis of chronic fatigue
                          syndrome by gene expression profiling
```

INVENTOR(S): Gow, John; Chaudhuri, Abhijit
PATENT ASSIGNEE(S): The University Court of the University of Glasgow, UK
SOURCE: PCT Int. Appl., 169pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006082390	A1	20060810	WO 2006-GB332	20060201

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: GB 2005-2042 A 20050201

AB Genes that show changes in levels of expression in chronic fatigue syndrome (myalgic encephalitis) are identified for use in the diagnosis of the disease and in its treatment. These genes include those encoding defensin .alpha.1, Hb .gamma., CXCR4, tubulin .beta.1, serine/threonine kinase 17B, HLA-DR.beta.4, and prostaglandin D2 synthase. There is a relatively small set of genes, identified as a hub set, that show changes in expression that result in changes in levels of expression of a no. of dependent or network genes. The genes identified provide objective disease markers that may be used in diagnostic tests to support the diagnosis of CFS/ME or for monitoring the effectiveness of therapy. They also provide a rational basis for classifying CFS/ME patients according to the biochem. lesion underlying their symptoms and enable provision of appropriate targeted therapies.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 2 OF 13 MEDLINE on STN
ACCESSION NUMBER: 2006331117 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16608858
TITLE: Phospholipase D1 regulates cell migration in a lipase activity-independent manner.
AUTHOR: Kim Jung Hwan; Kim Hyun-wook; Jeon Hyeona; Suh Pann-Ghill; Ryu Sung Ho
CORPORATE SOURCE: Division of Molecular and Life Sciences, Pohang University of Science and Technology, Pohang, 790-784, South Korea.
SOURCE: The Journal of biological chemistry, (2006 Jun 9) Vol. 281, No. 23, pp. 15747-56. Electronic Publication: 2006-04-10. Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200608
ENTRY DATE: Entered STN: 6 Jun 2006
Last Updated on STN: 24 Aug 2006
Entered Medline: 23 Aug 2006

AB Cell migration, a complex biological process, requires dynamic cytoskeletal remodeling. Phospholipase D (PLD) generates phosphatidic acid, a lipid second messenger. Although PLD

activity has been proposed to play a role in cytoskeletal rearrangement, the manner in which PLD participates in the rearrangement process remains obscure. In this study, by silencing endogenous PLD isozymes using small interfering RNA in HeLa cells, we demonstrate that endogenous PLD1 is required for the normal organization of the actin cytoskeleton, and, more importantly, for cell motility. PLD1 silencing in HeLa cells resulted in dramatic changes in cellular morphology, including the accumulation of stress fibers, as well as cell elongation and flattening, which appeared to be caused by an increased number of focal adhesions, which ultimately culminated in enhanced cell-substratum interactions. Accordingly, serum-induced cell migration was profoundly inhibited by PLD1-silencing. Moreover, the augmented cell substratum interaction and retarded cell migration induced by PLD1-silencing could be restored by the adding back not only of wild type, but also of lipase-inactive PLD1 into knockdown cells. Taken together, our results strongly suggest that endogenous PLD1 is a critical factor in the organization of the actin-based cytoskeleton, with regard to cell adhesion and migration. These effects of PLD1 appear to operate in a lipase activity-independent manner. We also discuss the regulation of Src family kinases by PLD1, as related to the modulation of Pyk2 and cell migration.

L22 ANSWER 3 OF 13 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:216606 HCAPLUS
DOCUMENT NUMBER: 142:292452
TITLE: Compns. and methods for treating and diagnosing chronic visceral hypersensitivity and irritable bowel syndrome, based on differential gene or protein expression
INVENTOR(S): Pasricha, Pankaj; Shenoy, Mohan; Winston, John
PATENT ASSIGNEE(S): Cytokine Pharmasciences, Inc., USA
SOURCE: PCT Int. Appl., 181 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005020902	A2	20050310	WO 2004-US27356	20040823
WO 2005020902	A3	20060727		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2005130189	A1	20050616	US 2004-923035	20040823

PRIORITY APPLN. INFO.: US 2003-496716P P 20030821

AB Compns. and methods for diagnosing and treating chronic visceral hypersensitivity (CVH) and CVH-assocd. disorders, such as irritable bowel syndrome, are disclosed. Genes differentially expressed in CVH tissues relative to normal tissues are identified. The genes and the gene products (i.e., the transcribed polynucleotides and polypeptides encoded by the genes) can be used as markers of CVH. The genes and the gene products can also be used to screen agents that modulate the gene expression or the activities of the gene products. The examples discuss the effects of acetic acid sensitization and CNI1493 treatment on the colon and S1 dorsal root ganglia in a rat model of visceral hypersensitivity. Gene expression profiles assocd. with these treatments

are presented, and rat CVH-related genes and polypeptides are identified.

L22 ANSWER 4 OF 13 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:838610 HCAPLUS
DOCUMENT NUMBER: 141:312238
TITLE: DNA microarray analysis of gene expression in the diagnosis of estrogen receptor positive- and negative-breast cancer
INVENTOR(S): Erlander, Mark G.; Ma, Xiao-Jun; Wang, Wei; Wittliff, James L.
PATENT ASSIGNEE(S): Arcturus Bioscience, Inc., USA
SOURCE: PCT Int. Appl., 226 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004079014	A2	20040916	WO 2002-XA2004006736	20040304
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2004079014	A2	20040916	WO 2004-US6736	20040304
WO 2004079014	A3	20050331		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

PRIORITY APPLN. INFO.: US 2003-451942P P 20030304
WO 2004-US6736 A 20040304

AB The invention relates to the identification and use of gene expression profiles, or patterns, suitable for identification of populations that are pos. and neg. for estrogen receptor expression. The gene expression profiles may be embodied in nucleic acid expression, protein expression, or other expression formats, and may be used in the study and/or diagnosis of cells and tissue in breast cancer as well as for the study and/or detn. of prognosis of a patient, including breast cancer survival.

L22 ANSWER 5 OF 13 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:808064 SCISEARCH
THE GENUINE ARTICLE: 850WP
TITLE: ADP-ribosylation factor6 regulates both [H-3]-noradrenaline and [C-14]-glutamate exocytosis through phosphatidylinositol 4,5-bisphosphate
AUTHOR: Zheng Q; Bobich J A (Reprint)
CORPORATE SOURCE: Texas Christian Univ, Dept Chem, Ft Worth, TX 76129 USA (Reprint)
j.bobich@tcu.edu
COUNTRY OF AUTHOR: USA
SOURCE: NEUROCHEMISTRY INTERNATIONAL, (OCT 2004) Vol. 45, No. 5,

pp. 633-640.
ISSN: 0197-0186.
PUBLISHER: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, ENGLAND.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 50
ENTRY DATE: Entered STN: 2 Oct 2004
Last Updated on STN: 2 Oct 2004
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB GTP phosphohydrolase (cell regulating) (EC 3.6.1.47, ADP-ribosylation factor6, ARF6) has been shown to play an important role in different steps of membrane trafficking. It also regulates chromaffin granule exocytosis through phosphatidylcholine phosphatidohydrolase (EC 3.1.4.14, PLD) activation. In this study, the role of ARF6 in neurotransmitter release from both dense-core granules (DCGs) and synaptic vesicles (SVs) in rat brain cortex nerve endings was investigated. We observed that synaptosomal ARF6 is largely particulate but moves to a less easily pelleted compartment upon nerve ending stimulation. We also found that direct inhibition of ARF6 by a specific antibody or interference with ARF6 downstream effects by a myristoylated N-terminal ARF6 peptide both significantly decreased both [H-3]-noradrenaline and [C-14]-glutamate exocytosis. Addition of phosphatidic acid (PA) and phosphatidylinositol 4,5-bisphosphate (PIP2) partially or completely restored exocytosis. These findings suggest that ARF6 plays important regulatory roles for both DCG and SV exocytosis by activating PLD and ATP:1-phosphatidyl-1D-myo-inositol 4-phosphate 5-phosphotransferase (EC 2.7.1.68, PI4P-5K) to enhance PIP2 synthesis and nerve ending membrane trafficking. (C) 2004 Elsevier Ltd. All rights reserved.

L22 ANSWER 6 OF 13 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:876816 SCISEARCH
THE GENUINE ARTICLE: 730HA
TITLE: Lung endothelial heparan sulfates mediate cationic peptide-induced barrier dysfunction: a new role for the glycocalyx
AUTHOR: Dull R O (Reprint); Dinavahi R; Schwartz L; Humphries D E; Berry D; Sasisekharan R; Garcia J G N
CORPORATE SOURCE: Univ Utah, Dept Anesthesiol, 3C-444 SOM, 30 No 1900 East, Salt Lake City, UT 84132 USA (Reprint); Johns Hopkins Sch Med, Dept Med, Div Pulm & Crit Care, Baltimore, MD 21287 USA; Dept Vet Affairs Med Ctr, Boston, MA 01230 USA; Harvard Univ, MIT, Div Hlth Sci & Technol, Cambridge, MA 02139 USA; Harvard Univ, MIT, Div Biol Engn, Cambridge, MA 02139 USA
COUNTRY OF AUTHOR: USA
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY-LUNG CELLULAR AND MOLECULAR PHYSIOLOGY, (1 NOV 2003) Vol. 285, No. 5, pp. L986-L995.
ISSN: 1040-0605.
PUBLISHER: AMER PHYSIOLOGICAL SOC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814 USA.
DOCUMENT TYPE: Editorial; Journal
LANGUAGE: English
REFERENCE COUNT: 42
ENTRY DATE: Entered STN: 24 Oct 2003
Last Updated on STN: 24 Oct 2003
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The endothelial glycocalyx is believed to play a major role in microvascular permeability. We tested the hypothesis that specific components of the glycocalyx, via cytoskeletal-mediated signaling, actively participate in barrier regulation. With the use of polymers of arginine and lysine as a model of neutrophil-derived inflammatory cationic proteins, we determined size- and dose-dependent responses of cultured bovine lung microvascular endothelial cell permeability as assessed by

transendothelial electrical resistance (TER). Polymers of arginine and lysine > 11 kDa produced maximal barrier dysfunction as demonstrated by a 70% decrease in TER. Monomers of L-arginine and L-lysine did not alter barrier function, suggesting a cross-linking requirement of cell surface "receptors". To test the hypothesis that glycosaminoglycans (GAGs) are candidate receptors for this response, we used highly selective enzymes to remove specific GAGs before polyarginine (PA) treatment and examined the effect on TER. Heparinase III attenuated PA-induced barrier dysfunction by 50%, whereas heparinase I had no effect. To link changes in barrier function with structural alterations, we examined actin organization and syndecan localization after PA. PA induced actin stress fiber formation and clustering of syndecan-1 and syndecan-4, which were significantly attenuated by heparinase III. PA-induced cytoskeletal rearrangement and barrier function did not involve myosin light chain kinase (MLCK) or p38 MAPK, as ML-7, a specific MLCK inhibitor, or SB-20358, a p38 MAPK inhibitor, did not alter PA-induced barrier dysfunction. In summary, lung endothelial cell heparan sulfate proteoglycans are key participants in inflammatory cationic peptide-induced signaling that links cytoskeletal reorganization with subsequent barrier dysfunction.

L22 ANSWER 7 OF 13 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2003:591251 SCISEARCH
THE GENUINE ARTICLE: 697VA
TITLE: Phosphatidic acid induces actin polymerization by activating protein kinases in soybean cells
AUTHOR: Lee S; Park J; Lee Y (Reprint)
CORPORATE SOURCE: Pohang Univ Sci & Technol, Div Mol & Life Sci, Pohang 790784, South Korea (Reprint)
COUNTRY OF AUTHOR: South Korea
SOURCE: MOLECULES AND CELLS, (30 JUN 2003) Vol. 15, No. 3, pp. 313-319.
ISSN: 1016-8478.
PUBLISHER: SPRINGER-VERLAG SINGAPORE PTE LTD, #04-01 CENCON I, 1 TANNERY RD, SINGAPORE 347719, SINGAPORE.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 42
ENTRY DATE: Entered STN: 25 Jul 2003
Last Updated on STN: 25 Jul 2003

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Phosphatidic acid (PA) levels rise in response to wounding, stress and elicitors, suggesting that it mediates defense responses in plants. During such responses, actin filaments are altered. Since PA induces actin polymerization in animal cells we examined its effect on actin structures in suspension-cultured soybean cells. PA caused a three to four fold increase in cells containing filamentous actin. Immunoblotting with anti-actin antibody showed that actin polymerized within 30 min of treatment. The effect of PA on actin polymerization appears to be mediated by protein kinases because: 1) the effect was suppressed by staurosporin, a general protein kinase inhibitor, and by the protein kinase C-specific inhibitor, calphostin, 2) calyculin A, an inhibitor of protein phosphatase I and 2A, mimicked the effect of PA on actin polymerization, and 3) PA activated protein kinases in soybean cells. We suggest that a 54 kDa Ca2+-dependent protein kinase may transduce the PA signal because EGTA inhibited the 54 kDa kinase and the PA-induced actin polymerization, and similar protein kinases have been reported to co-localize with and regulate actin filaments. Our results support the role of PA as a signal mediator and identify actin as a downstream target of PA.

L22 ANSWER 8 OF 13 HCAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 1

ACCESSION NUMBER: 2004:10817 HCAPLUS

DOCUMENT NUMBER: 140:249858
TITLE: Phosphatidylinositol 3-kinase-like activity in Tetrahymena. Effects of wortmannin and LY 294002
AUTHOR(S): Kovacs, Peter; Pallinger, Eva
CORPORATE SOURCE: Department of Genetics, Cell- and Immunobiology, Semmelweis University, Budapest, Hung.
SOURCE: Acta Protozoologica (2003), 42(4), 277-285
CODEN: ACPZAU; ISSN: 0065-1583
PUBLISHER: Nencki Institute of Experimental Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Insulin has many different effects on Tetrahymena, as e.g. on the glucose uptake, cell division, survival, phospholipase D activity and insulin prodn. Phosphatidylinositol (PI) 3-kinase is one of the key enzymes in the action of insulin. Thus it was supposed that similarly to the higher eukaryotes, PI 3-kinase activity plays fundamental role in the insulin action also in Tetrahymena. Here we report that PI 3-kinase-like activity is immunopptd. from Tetrahymena cell lysate with anti-IRS 1 and anti-p85 antibodies. Both immunoppts. contain higher PI 3-kinase activity from lysate of insulin-treated cells than the lysate of untreated ones. In vivo treatments with PI 3-kinase inhibitors wortmannin (100-500 nM) and LY 294002 (10-20 .mu.M) elevated the PI 3-kinase activity in the IRS 1-antibody precipitable material, while in anti-p85 antibody ppt. this activity was lower than in the controls. In vitro, wortmannin proved to be an effective PI 3-kinase inhibitor. Immunostaining revealed that p85 immunoreactivity localized to the cortex of cells, while IRS 1 localized cytoplasmically. In vivo treatments with both PI 3-kinase inhibitors elevated the amt. of IRS 1, while p85 immunoreactivity was increased only after wortmannin treatments. Both PI 3-kinase inhibitors reduced the F-actin content of cells. Wortmannin caused a forward cytoplasmic stream, which translocate the nucleus towards cytopharynx. These treatments inhibited the phagocytotic activity significantly. On the basis of the results, we propose that in Tetrahymena a PI 3-kinase-like activity is functioning; the ability of both PI 3-kinase inhibitors and insulin to influence the synthesis or assocn. of subunits of PI 3-kinase, and to influence F-actin remodeling and F-actin-dependent processes (e.g. phagocytosis) indicate the supposed activity of PI 3-kinase in Tetrahymena.

REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 9 OF 13 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:793791 SCISEARCH
THE GENUINE ARTICLE: 475ZU
TITLE: A 90-kD phospholipase D from tobacco binds to microtubules and the plasma membrane
AUTHOR: Gardiner J C; Harper J D I; Weerakoon N D; Collings D A; Ritchie S; Gilroy S; Cyr R J; Marc J (Reprint)
CORPORATE SOURCE: Univ Sydney, Sch Biol Sci, Macleay Bldg A12, Sydney, NSW 2006, Australia (Reprint); Univ Sydney, Sch Biol Sci, Sydney, NSW 2006, Australia; Penn State Univ, Dept Biol, University Pk, PA 16802 USA
COUNTRY OF AUTHOR: Australia; USA
SOURCE: PLANT CELL, (SEP 2001) Vol. 13, No. 9, pp. 2143-2158. ISSN: 1040-4651.
PUBLISHER: AMER SOC PLANT BIOLOGISTS, 15501 MONONA DRIVE, ROCKVILLE, MD 20855 USA.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 99
ENTRY DATE: Entered STN: 12 Oct 2001
Last Updated on STN: 12 Oct 2001

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The organization of microtubule arrays in the plant cell cortex

involves interactions with the plasma membrane, presumably through protein bridges. We have used immunochemistry and monoclonal antibody 6G5 against a candidate bridge protein, a 90-kD tubulin binding protein (p90) from tobacco BY-2 membranes, to characterize the protein and isolate the corresponding gene. Screening an Arabidopsis cDNA expression library with the antibody 6G5 produced a partial clone encoding phospholipase D (PLD), and a full-length gene was obtained by sequencing a corresponding expressed sequence tag clone. The predicted protein of 857 amino acids contains the active sites of a phospholipid-metabolizing enzyme and a Ca2+-dependent lipid binding domain and is identical to Arabidopsis PLD delta. Two amino acid sequences obtained by Edman degradation of the tobacco p90 are identical to corresponding segments of a PLD sequence from tobacco. Moreover, immunoprecipitation using the antibody 6G5 and tobacco BY-2 protein extracts gave significant PLD activity, and PLD activity of tobacco BY-2 membrane proteins was enriched 6.7-fold by tubulin-affinity chromatography. In a cosedimentation assay, p90 bound and decorated microtubules. In immunofluorescence microscopy of intact tobacco BY-2 cells or lysed protoplasts, p90 colocalized with cortical microtubules, and taxol-incluced microtubule bundling was accompanied by corresponding reorganization of p90. Labeling of p90 remained along the plasma membrane when microtubules were depolymerized, although detergent extraction abolished the labeling. Therefore, p90 is a specialized PLD that associates with membranes and microtubules, possibly conveying hormonal and environmental signals to the microtubule cytoskeleton.

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ACCESSION NUMBER: 2000:254111 SCISEARCH
THE GENUINE ARTICLE: 298GC
TITLE: Regulation of polymorphonuclear leukocyte phagocytosis by myosin light chain kinase after activation of mitogen-activated protein kinase
AUTHOR: Mansfield P J; Shayman J A; Boxer L A (Reprint)
CORPORATE SOURCE: Univ Michigan, Womens Hosp L2110, Dept Pediat, Box 0238, 1500 E Med Ctr Dr, Ann Arbor, MI 48109 USA (Reprint); Univ Michigan, Dept Pediat, Ann Arbor, MI 48109 USA; Univ Michigan, Dept Internal Med, Ann Arbor, MI 48109 USA
COUNTRY OF AUTHOR: USA
SOURCE: BLOOD, (1 APR 2000) Vol. 95, No. 7, pp. 2407-2412. ISSN: 0006-4971.
PUBLISHER: AMER SOC HEMATOLOGY, 1900 M STREET. NW SUITE 200, WASHINGTON, DC 20036 USA.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 54
ENTRY DATE: Entered STN: 2000
Last Updated on STN: 2000

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Polymorphonuclear leukocyte (PMNL) phagocytosis mediated by Fc gamma RII proceeds in concert with activation of the mitogen-activated protein (MAP) kinase, extracellular signal-regulated kinase ERK2, We hypothesized that myosin light chain kinase (MLCK) could be phosphorylated and activated by ERK, thereby linking the MAP kinase pathway to the activation of cytoskeletal components required for pseudopod formation. To explore this potential linkage, PMNLs were challenged with antibody -coated erythrocytes (EIgG). Peak MLCK activity, 3-fold increased over controls, occurred at 4 to 6 minutes, corresponding with the peak rate of target ingestion and ERK2 activity. The MLCK inhibitor ML-7 (10 mu mol/L) inhibited both phagocytosis and MLCK activity to basal values, thereby providing further support for the linkage between the functional response and the requirement for MLCK activation. The MAPK kinase (MEK) inhibitor PD098059 inhibited phagocytosis, MLCK activity, and ERK2 activity by 80% to 90%, To directly link ERK activation to MLCK activation, ERK2 was immunoprecipitated from PMNLs after EIgG ingestion. The isolated ERK2 was

incubated with PMNL cytosol as a source of unactivated MLCK and with MLCK substrate; under these conditions ERK2 activated MLCK, resulting in phosphorylation of the MLCK substrate or of the myosin light chain itself. Because MLCK activates myosin, we evaluated the effect of directly inhibiting myosin adenosine triphosphatase using 2,3-butanedione monoxime (BDM) and found that phagocytosis was inhibited by more than 90% but MLCK activity remained unaffected. These results are consistent with the interpretation that MEK activates ERK, ERK2 then activates MLCK, and MLCK activates myosin, MLCK activation is a critical step in the cytoskeletal changes resulting in pseudopod formation, (Blood, 2000;95:2407-2412) (C) 2000 by The American Society of Hematology.

L22 ANSWER 11 OF 13 MEDLINE on STN DUPLICATE 2

ACCESSION NUMBER: 1999241179 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10224668
TITLE: Phospholipases and phagocytosis: the role of phospholipid-derived second messengers in phagocytosis.
AUTHOR: Lennartz M R
CORPORATE SOURCE: Department of Physiology and Cell Biology, Albany Medical College, NY 12208, USA.. mlennartz@ccgateway.amc.edu
CONTRACT NUMBER: GM 45983 (NIGMS)
SOURCE: The international journal of biochemistry & cell biology, (1999 Mar-Apr) Vol. 31, No. 3-4, pp. 415-30. Ref: 108
Journal code: 9508482. ISSN: 1357-2725.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 18 Jun 1999
Last Updated on STN: 20 Apr 2002
Entered Medline: 8 Jun 1999

AB Phagocytosis, the process by which leukocytes recognize and destroy invading pathogens, is essential for host defense. The binding of foreign organisms to phagocytic leukocytes initiates a complex signaling cascade which ultimately results in the entrapment and destruction of the pathogen. The signal transduction pathway mediating phagocytosis is the subject of intense investigation and is known to include protein tyrosine kinases, GTP-binding proteins, protein kinase C (PKC), actin polymerization and membrane movement. A rapidly expanding body of evidence suggests that phospholipases play an integral role in phagocytosis by generating essential second messengers. Here we review the data linking activation of phospholipase A2 (PLA2), phospholipase C (PLC) phospholipase D (PLD), and phosphoinositide 3-OH kinase (PI(3)K) to antibody (IgG)-mediated phagocytosis. Evidence is presented that (1) PLA2-derived arachidonic acid (AA) stimulates NADPH oxidase and membrane redistribution during phagocytosis, (2) the inositol-3,4,5-triphosphate (IP3) and diacylglycerol (DAG) products of PLC activate NADPH oxidase and PKC, and (3) sequential activation of PLD and phosphatidic acid phosphohydrolase may provide an alternative pathway for generation of DAG. Additionally, considerable evidence exists that wortmannin, a PI(3)K inhibitor, depresses phagocytosis. This finding is discussed in the context of the extensive effects PI(3)K products have on endocytosis and exocytosis and the potential role of membrane redistribution in phagocytosis. Finally, a model is presented which integrates data obtained from a variety of phagocytic systems and illustrates potential interactions that may exist between phospholipase-derived second messengers and signaling events required for phagocytosis.

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ACCESSION NUMBER: 1998:626470 SCISEARCH
THE GENUINE ARTICLE: 111KR
TITLE: Localization of endogenous ARF6 to sites of cortical actin rearrangement and involvement of ARF6 in cell spreading
AUTHOR: Song J; Khachikian Z; Radhakrishna H; Donaldson J G (Reprint)
CORPORATE SOURCE: NHLBI, Cell Biol Lab, NIH, Bld 3, Room B1-22, Bethesda, MD 20892 USA (Reprint); NHLBI, Cell Biol Lab, NIH, Bethesda, MD 20892 USA
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF CELL SCIENCE, (AUG 1998) Vol. 111, Part 15, pp. 2257-2267.
ISSN: 0021-9533.
PUBLISHER: COMPANY OF BIOLOGISTS LTD, BIDDER BUILDING CAMBRIDGE COMMERCIAL PARK COWLEY RD, CAMBRIDGE CB4 4DL, CAMBS, ENGLAND.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 41
ENTRY DATE: Entered STN: 1998
Last Updated on STN: 1998

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB To study the function of the endogenous ARF6 GTP binding protein in cells, we generated an antibody which specifically recognizes ARF6, and not the other ARF proteins. Using this antibody, ARF6 was detected in all mouse organs tested and in a variety of cultured cell lines including RBL, MDCK, NRK, BHK, COS, and HeLa cells. In NRK cells, by immunofluorescence, ARF6 Localized to the plasma membrane, especially at regions exhibiting membrane ruffling, and was also concentrated in a fine punctate distribution in the juxtanuclear region. This pattern of localization of the endogenous protein was similar to the localization of ARF6 when overexpressed in NRK, or HeLa, cells. Treatments which perturb cortical actin in NRK cells, such as replating of cells after trypsinization or treatment with phorbol ester, resulted in the recruitment of endogenous ARF6 to the regions of cortical actin rearrangement. ARF6 activation and subsequent membrane recycling was required for cell spreading activity since expression of the dominant-negative, GTP-binding defective mutant of ARF6, T27N, previously shown to inhibit ARF6-regulated membrane recycling, inhibited cell attachment and spreading in HeLa cells. Furthermore, phorbol ester treatment enhanced the cell spreading activities in NRK cells, and in HeLa cells, but was not observed in cells expressing T27N, Taken together, these observations support a role for endogenous ARF6 in modeling the plasma membrane and cortical actin cytoskeleton.

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(FILE 'HOME' ENTERED AT 15:55:49 ON 30 JUL 2007)

FILE 'MEDLINE, HCAPLUS, BIOSIS, BIOTECHDS, SCISEARCH' ENTERED AT 15:56:21 ON 30 JUL 2007

L1 3312 S PHOSPHOLIPASE AND ACTIN
L2 1712 DUP REM L1 (1600 DUPLICATES REMOVED)
L3 145 S L2 AND ANTIBODY
L4 0 S L2 AND PHOSPHOLIPASE ANTIBODY
L5 0 S L2 AND PLD ANTIBODY
L6 0 S L2 AND PLD ANTIBODY
L7 0 S L2 AND PLD-ANTIBODY
L8 0 S L2 AND PHOSPHOLIPASE-ANTIBODY
L9 0 S ACTIN AND PHOSPHOLIPASE-ANTIBODY
L10 0 S ACTIN AND ANTI-PHOSPHOLIPASE-D
L11 0 S ANTI-PLD2 AND ACTIN

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     FILE 'REGISTRY' ENTERED AT 16:03:06 ON 30 JUL 2007
L12       25711 S ACTIN
L13         671 S BETA-ACTIN
L14           0 S BETA-ACTIN AND ANT--PHOSPHOLIPASE D

     FILE 'MEDLINE, HCAPLUS, BIOSIS, BIOTECHDS, SCISEARCH' ENTERED AT 16:04:59
     ON 30 JUL 2007
L15           0 S  ANTI-PLD2 AND ACTIN
L16           8 S ACTIN DETECTION AND ANTIBODY
L17           8 DUP REM L16 (0 DUPLICATES REMOVED)
L18           0 S PHOSLIPASE D AND ANTIBODY
L19         700 S PHOSPHOLIPASE D AND ANTIBODY
L20           1 S L19 AND BETA-ACTIN
L21          17 S L19 AND ACTIN
L22          13 DUP REM L21 (4 DUPLICATES REMOVED)

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